

[초청강연]

Health Risks Associated with Methylmercury Exposure and Molecular Toxicological Approaches for Its Study

Gi-Wook Hwang

Graduate School of Pharmaceutical Sciences, Tohoku University, Japan

The elemental form of mercury exists in liquid form at standard conditions for temperature and pressure. It is released from the earth's crust through degassing. It is also present in the environment as inorganic and organic compounds. Elemental mercury may be converted to inorganic compounds by oxidation and revert to elemental mercury by reduction. Inorganic mercury may become organic mercury through the action of certain anaerobic bacteria, and degrade to inorganic mercury slowly. Recently, mercury emission and release into the environment have been increasing by human activities including coal combustion and artisanal small-scale gold mining. An international convention of mercury is prepared to reduce mercury usage and load to the environment and to prevent the pollution and health damage.

Recent epidemiological studies have suggested excessive intake of methylmercury during pregnancy to have an adverse effect on the fetal neural development. However, the molecular mechanisms underlying methylmercury-induced neurotoxicity are still only poorly understood. We have identified several factors involved in methylmercury toxicity using multiple omics techniques such as genomic, transcriptomics, proteomics, and metabolomics. In this presentation, I will introduce health risks associated with methylmercury exposure and functional factors suggested to be involved in its toxicity, and would like to discuss our findings regarding their roles

References

- Hwang, G. W., Fukumitsu, T., Ogiwara, Y., Takahashi, T., Miura, N., Kuge, S., Naganuma, A., 2016, Whi2 enhances methylmercury toxicity in yeast via inhibition of Akr1 palmitoyltransferase activity, *Biochim. Biophys. Acta.*, 1860, 1326-1333.
- Iwai-Shimada, M., Takahashi, T., Kim, M. S., Fujimura, M. Ito, H., Toyama, T., Naganuma, A., Hwang, G. W., 2016, Methylmercury induces the expression of TNF- α selectively in the brain of mice, *Sci. Rep.*, 6, 38294.
- Kim, M. S., Takahashi, T., Lee, J. Y., Toyama, T., Hoshi, T., Kuge, S., Fujiwara, Y., Naganuma, A., Hwang, G. W., 2019, Methylmercury induces the expression of chemokine CCL4 via SRF activation in C17.2 mouse neural stem cells, *Sci. Rep.*, 9, 4631.
- Lee, J. Y., Ishida, Y., Takahashi, T., Naganuma, A., Hwang, G. W., 2016, Transport of pyruvate into mitochondria is involved in methylmercury toxicity, *Sci. Rep.*, 6, 21518.
- Sato, M., Toyama, T., Lee, J. Y., Miura, N., Naganuma, A., Hwang, G. W., 2018, Activation of ornithine decarboxylase protects against methylmercury toxicity by increasing putrescine, *Toxicol. Appl. Pharmacol.*, 356, 120-126.
- Takahashi, T., Kim, M. S., Iwai-Shimada, M., Hoshi, T., Fujimura, M., Toyama, T., Fujiwara, Y., Naganuma, A., Hwang, G. W., 2019, Induction of chemokine CCL3 by NF- κ B reduces methylmercury toxicity in C17.2 mouse neural stem cells, *Environ. Toxicol. Phar.*, in press.
- Zhang, Z. T., Ogiwara, Y., Ito, Y., Hikida, A., Miura, N., Kuge, S., Naganuma, A., Hwang, G. W., 2017, Akr1 attenuates methylmercury toxicity through the palmitoylation of Meh1 as a subunit of the yeast EGO complex, *Biochim. Biophys. Acta.*, 1861, 1729-1736.